IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Patent Application of	Group Art Unit: 1642
Hilmar Meek Warenius	Examiner: Mark Halvorson
Application No.: 10/508,873	Confirmation No.: 1109
Filed: December 6, 2004	
For: TREATING CANCER	

DECLARATION UNDER 37 C.F.R. § 1.132

Sir:

1. I, Hilmar Meek Warenius, am a co-inventor of this patent application.

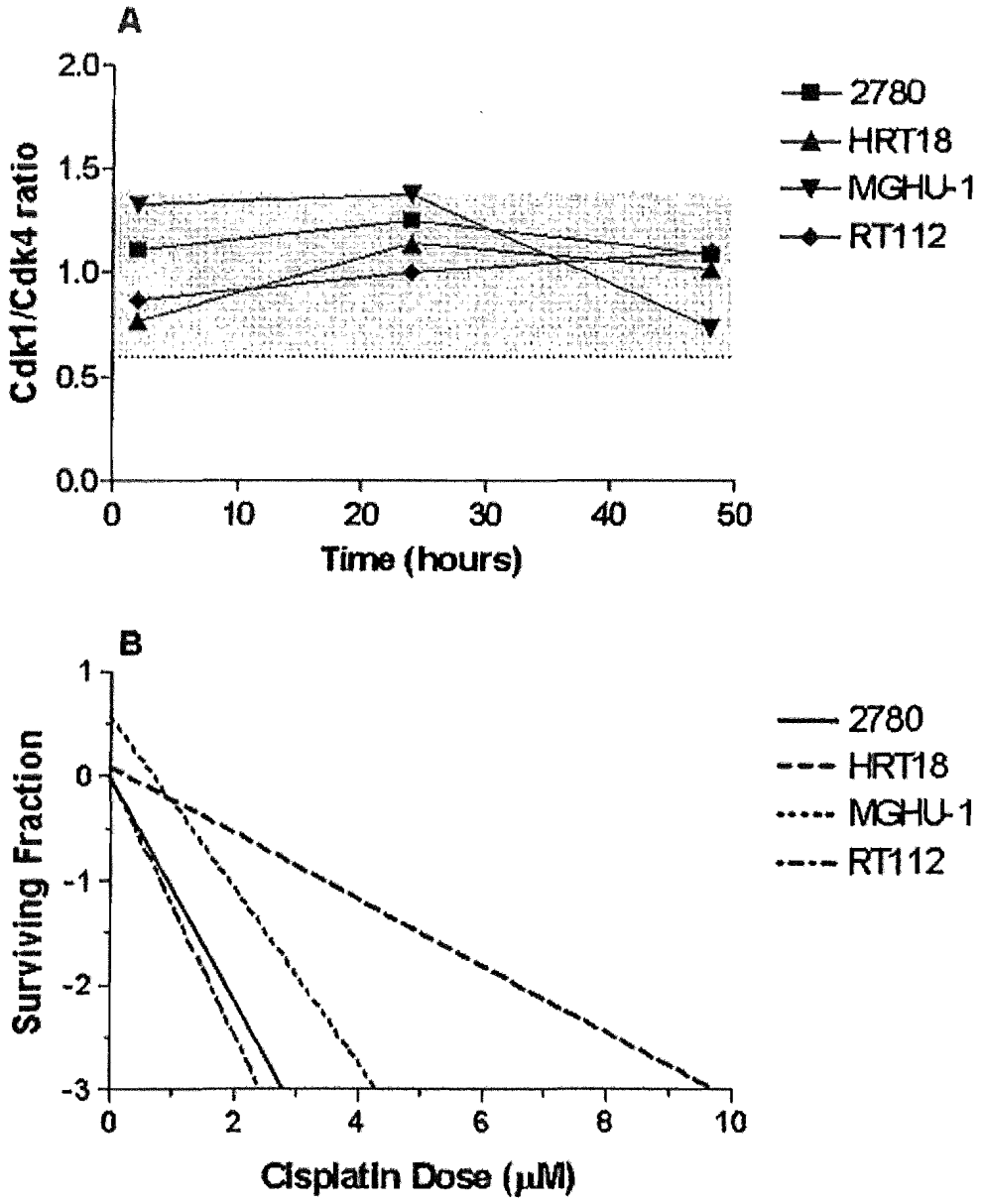
2. I am an expert in the fields cancer research and therapy. My Curriculum Vitae is attached as documentation of my credentials (see, e.g., Exhibit A).

3. I have read the specification and the file history, including the past and the present outstanding office actions and I believe I understand the issues presented by the Patent Office in the outstanding office action regarding the pending claims of the application (which will be referred to hereinafter as "the/this invention").

4. It is the Patent Office's position that claims 1, 2, 5, 7 and 8 are allegedly unpatentable over Hybridon (WO 99/27087) alone or in further view of TheRyte Limited (WO 99/42821). It is the Patent Office's position that the foregoing references allegedly teach and suggest a ratio change of CDK1 and CDK4. The Patent Office appears to consider that the Hybridon reference teaches that the antisense molecules used as a potential cancer therapy described therein would inhibit the expression of CDK4 and would therefore alter the ratio of the CDK1 and CDK4 gene products.

5. To address this issue, as an expert in this field and as co-inventor of this invention I provide the following data demonstrating that this position is in error.

6. First, Hybridon does not mention CDK1, and certainly provides no teachings in relation to what happens to the levels of CDK1 on administration of the antisense agent. For example, the concentration of CDK1 may decrease by the same amount as the concentration of CDK4 on administration of the antisense agent, leading to no change in the ratio of the levels of these two gene products as set forth by the presently claimed invention, this is supported by the fact that other chemotherapeutic agents do not alter the ratio of CDK1 and CDK4. In support of this position the Examiner is directed to Figure 1A below which shows the effect at 2, 24 and 48 hours on the CDK1/CDK4 ratio of the well known anticancer agent cis-diamminedichloroplatinum (CDDP) on 2780, HRT18, MGHU-1 and RT112 cancer cell lines, each treated at a CDPP dose which reduces cell survival to 10%. Furthermore, Figure 1B shows that CDDP is effective in killing the cancer cells. It can be seen that CDDP is an effective chemotherapeutic agent, but does not alter the ratio of CDK1 to CDK4 gene products significantly, and the ratio remains within the range of 0.6 to 1.6 throughout the course of the experiment (see also Warenius et al. Anticancer Research, vol. 29 no. 6, pp. 1933-1941, June 1, 2009). Thus it is clear that not all chemotherapeutic agents alter the ratio of CDK1 to CDK4 gene products and thus the alleged teaching relied upon by the Patent Office cannot be derived from the Hybridon reference.



7. The Patent Office also appears to allege that nowhere in the specification is it demonstrated that agents that are effective in the treatment of cancer alter the ratio of CDK1 and CDK4 gene products. Figure 2 (below) shows Western Blots from Cdk1 and Cdk4 in protein lysates of RT112 bladder cancer cells. Western blotting is carried out by the method described in PCT/GB99/00506. The top panel shows a progressive increase in Cdk1 protein with time in the presence of the peptide agent PRGPRP, while in the presence of the peptide PRRPGP the amount of Cdk1 protein remains relatively constant. The bottom panel shows that

the quantity of Cdk4 does not change significantly over time in the presence of either peptide. Thus it can be seen that in the presence of PRGPRP the Cdk1/Cdk4 ratio increases while in the presence of PRRPGP there is no significant change in the ratio. Figure 3 demonstrates that RT112 bladder cancer cells are killed by PRGPRP but not by PRRPGP, evidencing that an effective agent can be identified simply by determining whether the ratio of Cdk1 to Cdk4 has been altered.

FIGURE 2

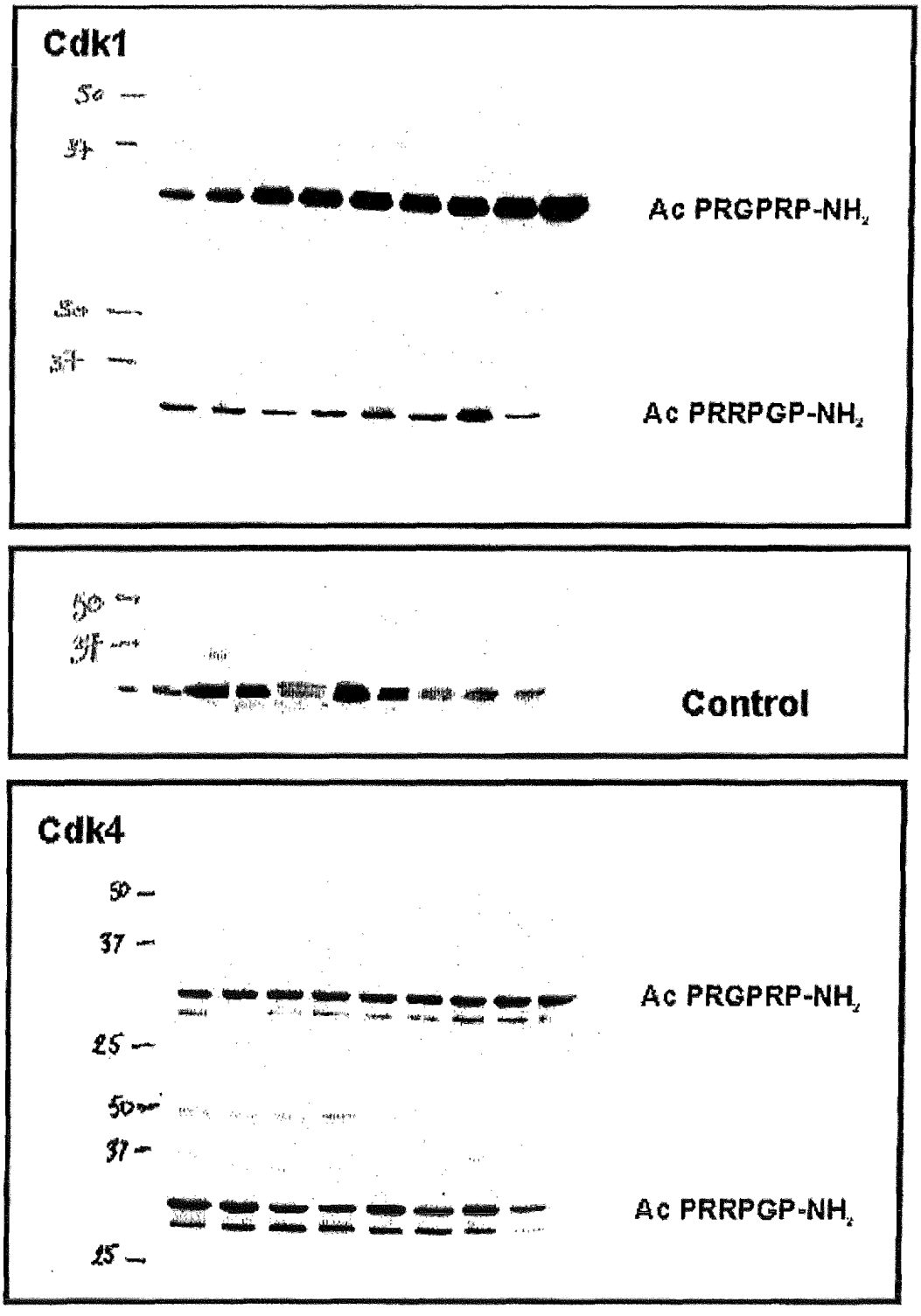
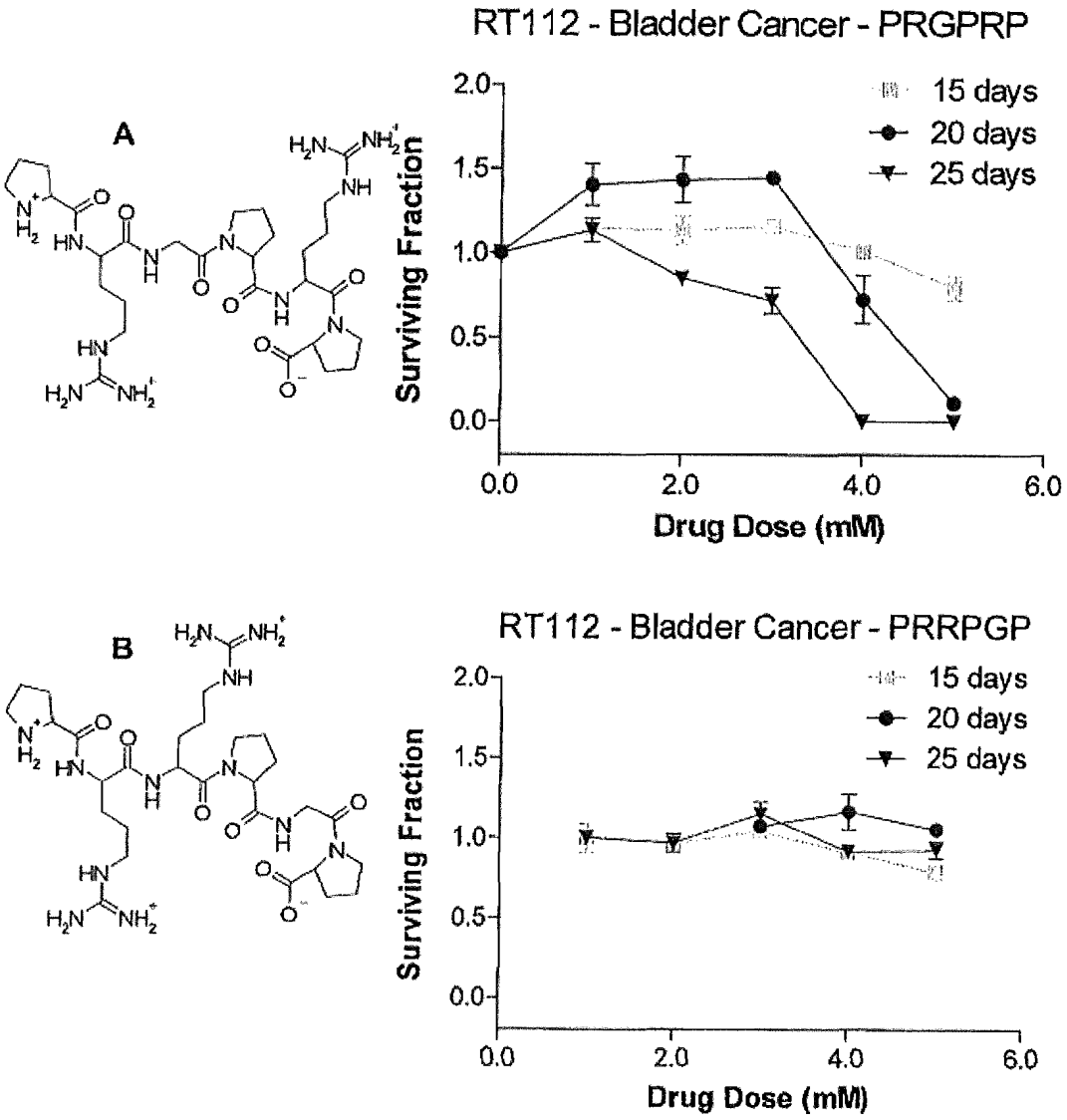

Cdk1
Ac PRGPRP-NH2
Ac PRRPGP-NH2
Control
Cdk4
50
37
25
Ac PRGPRP-NH2
Ac PRRPGP-NH2

FIGURE 3



8. Accordingly, based upon the foregoing evidence a chemotherapeutic agent does not necessarily result in a change in the ratio of Cdk1 and Cdk4 as alleged by the Patent Office with reference to Hybridon; thus the teachings or

suggestions relied upon by the Patent Office are not supported by the reference. Furthermore, the evidence demonstrates that an agent that changes the ratio of Cdk1 and Cdk4 is effective at killing cells having cell proliferative disorders (e.g., cancer cells) and that identifying such agents is useful for development of chemotherapeutics.

9. I hereby declare that all statement made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Respectfully Submitted

Date: June 14th 2010

Hilmar Meek Warenius

EXHIBIT A

CURRICULUM VITAE

Professor Hilmar M Warenius
MA MB BChir DMRT PhD FRCR FRCP

CEO HilRos Ltd

Visiting Professor in Anti-cancer Drug Development,
University of Southampton, School of Chemistry

Name	Hilmar Meek WARENIUS		
Address	14 Delavor Road Heswall Wirral, Merseyside Tel: 051 342 3034		
Education	Penzance Grammar School		
	Downing College, Cambridge		
	The Middlesex Hospital Medical School		
Qualifications	MA Nat Sci Tripos University of Cambridge	1967	
	Member of the Royal College of Surgeons, Licentiate of the Royal College of Physicians Examining Board		1968
	MB BChir University of Cambridge	1968	
	Member of the Royal College of Physicians	1971	
	Diploma in Medical Radiation Therapy Examining Board		1973
	Fellow of the Royal College of Radiologists		1975
	PhD University of Cambridge	1980	
	Fellow of the Royal College of Physicians		1986
	Cardiff University Expert Witness		2005

PRESENT POSITION

CEO HilRos Ltd

OTHER APPOINTMENTS
October 2006 – October 2009
Visiting Professor in Anti-cancer Drug Development, University of Southampton, School of Chemistry.

Honorary Professor Guanxi Medical University, Nanning, China.

POSTS PREVIOUSLY HELD
November 1998 – March 2009

Chief Medical Officer and Director of R & D
TheRyte Ltd

1997 - 2006
University of Liverpool Professor of Oncology Research
Oncology Research Unit
School of Clinical Sciences
The University of Liverpool
LIVERPOOL L69 3GA

August 1990 - 1997
University of Liverpool Cancer Research Campaign Professor of Oncology Research
CRC Oncology Research Unit
Department of Medicine
University of Liverpool
LIVERPOOL L69 3BX

October 1982 - August 1990
Cancer Research Campaign Professor of Radiation Oncology
Medical Research Council Honorary Trials Coordinator
Honorary Consultant Radiotherapist and Oncologist
Mersey Regional Centre for Radiotherapy & Oncology and Douglas Cyclotron
Centre, Clatterbridge Hospital, Bebington, Wirral

August 1980 - September 1982 Consultant Radiotherapist and Oncologist
Honorary University Lecturer.
Regional Radiotherapy Centre
Newcastle General Hospital, Westgate Road, Newcastle Upon Tyne.

April 1979 - August 1980 University first Assistant Radiotherapist,
Honorary Senior Registrar in Radiotherapy
Regional Radiotherapy Centre, Newcastle General Hospital, Newcastle Upon Tyne

October 1975 - April 1979 Medical Research Council Research Fellow.
MRC Clinical Oncology and Radiotherapeutics Unit
New Addenbrooke's Hospital, Cambridge

March 1974 - October 1975 University First Assistant Radiotherapist,
Honorary Senior Registrar in Radiotherapy to Professor J.S. Mitchell
New Addenbrooke's Hospital, Cambridge

April 1972 - March 1974 Registrar in Radiotherapy to Miss M.D. Snelling, Dr A.M. Jelliffe, Professor N.M. Bleehen and Dr M.F. Spittle
Meyerstein Institute of Radiotherapy, The Middlesex Hospital, London
September 1971 - April 1972 Research Assistant to Professor N.M. Bleehen
Academic Department of Radiotherapy
Middlesex Hospital Medical School, London

February - September 1971 Casualty Medical Officer:
Consultant in Charge : *Dr P.A.J. Ball*
The Middlesex Hospital, London

July 1970 - February 1971 Senior House Officer in Radiotherapy to Dr R Morrison

Hammersmith Hospital, London

September 1969 - July 1970 Senior House Officer in Medicine to Professor Sir David W. Smithers, and Professor Gordon Hamilton-Fairley
The Royal Marsden Hospital, Sutton, Surrey

CLINICAL EXPERIENCE
I have had a wide experience of treating malignancies with both conventional radiotherapy, high LET fast neutron therapy and chemotherapy. My experience has covered a wide range of tumours at most anatomical sites except for leukaemias. My special interests have been in breast cancer, lung cancer and gynaecological cancer. In addition I am particularly concerned with the development of good clinical communication skills both for oncologists and other medical and para-medical practitioners.

TEACHING EXPERIENCE
In addition to teaching and testing general clinical medical skills with undergraduates in the department of medicine, I have made an active contribution to the development of the Liverpool new medical undergraduate curriculum by writing study guide outlines and leading the development by myself and colleagues of an oncology (the first definitive) study guide. I have participated in the production of the computerised oncology teaching module "LETSGO" and I have also undertaken to act as a group facilitator for the first batch of students to start the new curriculum in October 1996. I have a particular interest in the teaching of communication skills and have undergone training in both carrying out effective communication and teaching others these skills under Dr Peter McGuire and Dr Leslie Fallowfield. I regularly work as a communications skill teacher on courses for medical undergraduates both in Liverpool and Cambridge, for nurses on Liverpool postgraduate courses and for qualified medical practitioners on short residential courses. I also teach on molecular and cell biological aspects of gene expression particularly with regard to the control of cell division to medical undergraduates on the B Clin Sci course and to nurses taking the Liverpool degree course.

MEDICO-LEGAL EXPRIENCE
As a Medico-legal expert witness in the field of Oncology I have advised on over 200 cases, my principal contribution being in the area of causation. I am a founder member of the Expert Witnesses Institute and a Cardiff University Accredited Expert Witness by examination.

I was invited to write book as contribution to the Medico-Legal Practitioner Series entitled: "ONCOLOGY, HILMAR M WARENIUS" first published in Great Britain 1998 by Cavendish Publishing Ltd. LONDON.

I have been an invited speaker on two occasions recently at conferences for Lawyers organised by 'Action for Victims of Medical Accidents' and the 'Medico-Legal Practitioners Association'.

SCIENTIFIC EXPERIENCE
My earliest scientific work involved the study of xenograft model systems for the investigation of the efficacy of chemotherapy in human cancer for which I was awarded the degree of PhD by the University of Cambridge in 1980. In addition to investigating prodrug therapy with the alkylating agent aniline mustard, I carried out, in collaboration with Professor Cesar Milstein, the very first experiments to test whether monoclonal antibodies

could could be used to selectively localise radioactivity in human cancer using HT29 human colon cancer xenografts.

It has become increasingly apparent over the past decade that established in-vitro cultures of human cancer cells show a similar range of responses to radiation and cytotoxic drugs to that seen in the clinic. I have carried out a body of work establishing the response patterns of a wide range of such human cancers cells to both low LET (conventional) radiation and high LET neutrons as well as to chemotherapeutic agents.

Subsequently, I moved to investigating how the expression of proteins controlling cancer cell growth survival and death may be related to the differences in sensitivity which individual cancers show to these treatments.

During this period I began to realise that, as a result of immortalisation, the complex interactive systems controlling division, apoptosis, differentiation and senescence in normal cells become markedly destabilised. This destabilisation would be exacerbated by ongoing genetic instability, which generates multiple evolving cancer cell phenotypes, each of which can be considered as a unique complex emergent system.

I have suggested that following immortalisation, stabilisation (neostasis) becomes critically important for successful survival and termed the genes responsible for neostasis, Critical Normal Genes because their function is critical to neoplastic cell survival (Warenius, HM, Anticancer Research 22: 2651-2656 (2002)).

The above concepts led to a realisation that a highly significant co-relationship between the expression of CDK1 and CDK4 found across all cancer cell lines examined in-vitro and also in clinical malignant melanoma, could mean that these proteins were acting as Critical Normal Gene Products. This insight was strengthened by the finding that mice in which CDK4 expression was completely knocked out did not get cancer. Transfection experiments in my laboratories indicated that CDK1 levels were controlled by CDK4 but not through the classical kinase mechanism of the latter, leading to collaborative studies with Professor Jeremy Kilburn and Professor Jon Essex at the University of Southampton, Department of Chemistry which detected a unique novel partially hydrophobic short amino acid sequence in the non-kinase region of CDK4. The central hexameric peptide from this region has been shown to selectively kill cancer but not normal cells in a previously undescribed manner.

Based on the above findings, I am now developing novel anti-cancer agents at the University of Southampton which have improved cell uptake and target binding.

PATENT PORTFOLIO
Title: Treating Cancer (General)
Our ref: T1
Granted in: PCT, Great Britain, Australia, USA
Filed in: Japan, Hong Kong

Title: Treating Cancer (CDK1/CDK4)
Our ref: T2
Granted in: PCT, Europe, Australia, USA, Austria, Belgium, Switzerland, Germany, France, Great Britain, Italy
Filed in: Japan

Title: Treating Cancer (p53/Cyclin D1)
Our ref: T3
Granted in: PCT, Europe, Germany, France, Great Britain, Italy
Filed in: Japan

Title: Treating Cancer (p21/Cyclin D1)
Our ref: T4
Granted in: PCT, Europe, Germany, France, Great Britain, Italy
Filed in: Japan

Title: Treating Cancer (p21/Raf-1)
Our ref: T5
Granted in: PCT, Europe, Germany, France, Great Britain, Italy
Filed in: Japan

Title: Treating Cancer (Taxol/p21)
Our ref: T6
Granted in: PCT, Australia
Filed in: Japan

Title: Treating Cancer (Taxol/p53)
Our ref: T7
Granted in: PCT
Filed in: Japan

Title: Delivery System (TheraSol)
Our ref: T8
Granted in: PCT, USA
Filed in: Japan, Europe, Hong Kong

Title: Treating Cancer (Critical Normal Gene Products)
Our ref: T12
Filed in: PCT, USA
Title: Treating Cancer (Decapeptide)
Our ref: T13
Filed in: PCT, Great Britain

Title: Treating Cancer
Our ref: T14
Filed in: Great Britain, September 2006

MOST RECENT PUBLICATIONS

1. Britten, R.A., **Warenius, H.M.**, Parkins, C. and Peacock, J.H.
The Inherent Cellular Sensitivity to 62.5 MeV (p-Be+) Neutrons in Human Cells of Different Photon Sensitivity.
Int J Rad Biol 61, 805-812 (1992)

2. Husband, D.J., Errington, R.D., Myint, S., Littler, J.A.H. and **Warenius, H.M.**
Accelerated fast neutron therapy: a pilot study
Brit J Radiol 65, 691-696, (1992)

3. Britten, R.A., Peacock, J. and **Warenius H.M.**
Collateral Resistance to Photon and Neutron Irradiation is Associated with Acquired Cis-Platinum Resistance in Human Ovarian Tumour Cells.
Radiotherapy and Oncology 23, 170-175 (1992)

4. Britten, R.A., Green, J.A and **Warenius, H.M.**
Cellular Glutathione (GSH) and Glutathione S-Transferase (GST) Activity in Human Ovarian Tumour Biopsies Following Exposure to Alkylating Agents.
Int J Rad Onc Biol Phys 24, 527-53 (1992)

5. Britten, R.A., **Warenius, H.M.**, Masters, R.W. and Peacock, J.H.
The Differential Induction of Collateral Resistance to 62.5 MeV (p->Be+) Neutrons and 4 MeV Photons by Exposure to Cisplatinum.
Int J Rad Oncol Biol Phys 26, 837 - 843 (1993)

6. Britten, R.A. and **Warenius, H.M.**
De novo cisplatinum resistance does not influence cellular radiosensitivity.
Eur J Cancer 29A, 1315 - 1320 (1993)

7. Pollock, J.M., Rowan, T.G., Dixon, J.B., Carter, S.D., Spiller, D. and **Warenius, H.M.**
Alteration of cellular immune responses by nutrition and weaning in calves.
Res Vet Sci 55, 298-305 (1993)

8. **Warenius, H.M.**
Fast neutron therapy. The UK experience.
Acta Oncologica, Vol 33, No 3, 289-292 (1994)

9. **Warenius, H.M.**, Britten, R.A. and Peacock, J.
The relative cellular radiosensitivity of 30 human in vitro cell lines of different histological type to High LET 62.5 MeV (p->Be+) fast neutrons and 4 MeV photons.
Radiotherapy and Oncol, 30, 83-89 (1994)

10. **Warenius, H.M.**, Browning, P.G., Morton, I.E., Britten, R.A. and Peacock, J.H.
Identification of human in vitro cell lines with greater intrinsic cellular radiosensitivity to 62.5 MeV (p->Be+) neutrons than 4 MeV photons.
Int J Rad Oncol Biol Phys, Vol 28, No 4, 913-920 (1994)

11. **Warenius, H.M.**, Browning, P.G.W., Britten, R.A., Peacock, J.H. and Rapp, U.R.
C-raf-1 proto-oncogene expression relates to radiosensitivity rather than radioresistance.
Europ J Cancer, Vol 30A, No 3, 369-375 (1994)

12. **Warenius, H.M.** and Britten, R.A.
In vitro studies of intrinsic cellular radiosensitivity following 4 MeV photons or 62.5 MeV (p->Be+) neutrons.
Acta Oncologica, Vol 33, No 3, 241-249 (1994)

13. Carsberg, C.J., **Warenius, H.M.** and Friedmann, P.S.

Combined RAF1 protein expression and p53 mutational status provides a strong predictor of cellular radiosensitivity.
Brit J Cancer, 83, 8, 1084-1095 (2000)

25. **Warenius, H.M.**
Are Critical Normal Gene Products In Cancer Cells The Real Therapeutic Targets?
Anti-Cancer Research, 22: 2651-2656 (2002)

26. Seabra L, **Warenius H**. Proteomic co-expression of cyclin-dependent kinases 1 and 4 in human cancer cells. *Eur J Cancer* 2007; **43**: 1483 – 1492.

27. **Warenius H**, Howarth A, Seabra L, et al. Dynamic heterogeneity of proteomic expression in human cancer cells does not affect Cdk1/Cdk4 co-expression. J Exp Ther Oncol 2008;7:237–254.

28. **Warenius H**, Kyritsi L, Grierson I, et al. Spontaneous regression of human cancer cells *in-vitro*: Potential role of disruption of Cdk1/Cdk4 co-expression. Anticancer Res 2009;29:1933–1942.

29. Warenius H, Seabra L, Kyritsi L, et al. Theranostic proteomic profiling of cyclins, cyclin dependent kinases and Ras in human cancer cell lines is dependent on p53 mutational status. Int J Onc. 2008;32:895-907.

30. Warenius H. Technological challenges of theranostics in oncology. Expert Opinion on Medical Diagnostics 2009; 3: 381-393

ABSTRACTS

1. **Warenius, H. M.** and Seabra, L.A.
Co-expression of cdc2 and cdk4 in human cancer cells is disrupted following g-irradiation.
Proceedings of the American Society for Therapeutic Radiology & Oncology, 37th Annual Meeting, Miami.
Int J Rad Oncol Biol Phys, 32, Supp 1, 235 (1995)

2. **Warenius, H.M.** and Jones, B.
Suggestions for fast neutron clinical schedules using fractions of less than 1.0Gy.
XVIIth AROI Conference, Lucknow, India, December 1995.

3. **Warenius, H.M.**, Gorman, T., Barraclough, R., Thompson, C., Jones, M. and Rudland, P.
Post-radiation exit from G2 is related to endogenous raf-1 protein levels in human cells expression wild-type p53.
BOA 1996 Joint Meeting, July 1996.

4. **Warenius, H.M.**, Gorman, T., Barraclough, R., Thompson, C., Jones, M. and Rudland, P.
Post-radiation exit from G2 is related to endogenous raf-1 protein levels in human cells expressing wild-type p53.
ASTRO, Los Angeles, USA, October 1996.

5. Seabra, L.A. and **Warenius, H.M.**
Wild type CDK1 and CDK4 proteins are co-elevated in human cancer.
AACR, New Orleans, USA, March 1998.

6. Moumtzi, S. and **Warenius, H.M.**

Cytochalasin B-induced polykaron formation as an indicator of proliferation in p53 dominant-negative transformation of early passage human skin fibroblasts.
AACR, Philadelphia, USA, April 1999.

7. Xiao, Q. and **Warenius, H.M.**
The Cytological Effects Of Ionizing Radiation On Human Cancer Cells Of Different Histogenetic Origin.
AACR, San Francisco, March 2000

8. Jones, M. and **Warenius, H.M.**
Predictive Relationships Between C-Raf 1, p53 Mutational Status, G2+M Exit and Radiosensitivity Are Found At Low But Not High Doses Of γ-Radiation.
New Orleans, March, 2001

9. **Warenius, H.M.**, Seabra, L., Kyritsi, L., Jones, M., Thomas, C., White, R. and Howarth, A.
Proteomic Co-Expression Patterns Of Critical Normal Genes Are Found In Human Cancer Cell Lines But Not In Normal Cells.
BCRM, Manchester, June 2004.

10. **Warenius, H.M.,** Kilburn, J.D., Essex, J.W., Thomas, C., Howarth, A., Vasireddy, L.
A CDK4 Homologous Peptide With A New Mode Of Selective Cancer Cell Killing.
AACR NCI EORTC International Conference Molecular Targets and Cancer Therapeutics: Discovery, Biology and Clinical Applications Conference, Philadelphia, November 14-18 2005.

INVITED PAPERS
11-13 June 1990.British Institute of Radiology Radiobiology Proffered Papers, London.
Peacock, J., Parkins, C.S., Steel, G.G., Britten, R.A. and Warenius, H.M.
"The effect of LET on the radiation response of human tumour cell lines of differing radiosensitivity."
23 February 1990.

British Association for Cancer Research
Tidd, D.M., Spiller, D.G., Goodwin, C.C. and Warenius, H.M.
"Properties of c-myc methylphosphonodiester/phosphodiester chimeric antisense oligonucleotides."
March 1990

ICRR, Toronto, Canada
"Variation of cellular sensitivity to 62.5MeV (p->Be+) neutrons in human cells."
July 1991

EACR, Genoa, Italy
"Acquired resistance to cisplatinum in human in-vitro cell lines correlates with resistance to photons and neutrons. Intrinsic resistance does not". October 1991

Association of Radiation Research
"Acquired resistance to cisplatinum correlates more with cross resistance to neutrons than photons in 5 human in vitro cell lines."
April 1992

October 1994

Royal Marsden Hospital, Sutton
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
December 1994

University of Uppsala, Sweden
a) PhD opponent "Biological effects of accelerated ions".
b) "The relationship of therapeutic response and the expression of genes related to cellular proliferation".
April 1995

Harvard Medical School, Massachusetts General Hospital, Boston, USA
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
September 1995

Fox Chase Cancer Center, Philadelphia, USA
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
September 1995

University of Pennsylvania, Department of Radiation Oncology, USA.
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
September 1995

MD Anderson Cancer Center, Houston, USA
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
September 1995

Stanford University, San Francisco, USA
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
September 1995

Wayne State University, Detroit, USA
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
October 1995

The Institute for Molecular Biology & Biotechnology, McMaster University, Hamilton, Ontario, Canada
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
October 1995

Alder Hey Hospital, Liverpool
"Molecular Biology of cell division. Is it clinically relevant?"
October 1995

Institute of Cancer Research Calcutta, India
"The relationship of therapeutic response and the expression of genes related to cellular proliferation".
January 1996

BACR Edinburgh, Flow Cytometry workshop
"The molecular control of the cell cycle"
April 1996

Institute for Cancer Studies, University of Sheffield
'The relationship of therapeutic response to the expression of genes controlling cellular proliferation,' October 1996

Institute of Environmental and Biological Sciences, Lancaster University
'The role of cell cycle control molecules in response to genotoxic agents.'
October 1997

PRESENTATIONS
Innovative Drug Development 1999
New York USA, April 1999.

Genomic Partnering Europe
Munich, Germany, May 1999.

SUPERVISED POSTGRADUATE DEGREES:
Paul Browning, PhD, 'Proto-oncogene expression and intrinsic cellular radiosensitivity', September 1996

Matthew Jones, PhD, 'Effects of radiation on the G2/M checkpoint in human tumour cells of differing radiosensitivities', May 1997

Laurence Seabra, PhD, 'Molecular cell cycle control and therapeutic response in human cancer cells', May 1999

Xiao Qiang, MD, 'The Cytological Effects of Ionising Radiation On Human Cancer Cells of Different Histogenetic Origin', June 2000

Mark Jones, 'The Predictive Relationship Between Raf-1 Protein Level, Radiosensitivity , And Post-Irradiative G_2+M Cell Cycle Accumulation Influenced By Wild Type P53', Submitted January 2006

LEARNED SOCIETIES
Fellow of the Royal College of Physicians.
Fellow of the Royal College of Radiologists.
Member of The American Association For Cancer Research
British Institute of Radiology.
British Association for Cancer Research.
British Society of Immunology.
British Oncological Association.
Expert Witnesses Institute (Founder member).

REFEREE FOR JOURNALS:-

British Journal of Cancer.
British Journal of Radiology.
European Journal of Cancer.
International Journal of Radiation Biology

OTHER ACTIVITIES Swimming, Blues and 1950s Rock Guitar, Cooking, St Peter's Church Choir, Book Club, Building a 1942 Model of HMS Belfast from mahogany.